THE RECOVERY KERNEL

DISCONTINUATION PROTOCOL: METHAMPHETAMINE

The Crash, the Timeline, and the Road Back to a Working Dopamine System.

This guide follows the same structure as the Recovery Kernel's Forced Discontinuance series — a safety framework, a genetic cross-reference, a use-pattern classification, a recovery protocol, and a full protocol map — adapted to what methamphetamine discontinuation actually is. It is not a seizure risk the way benzodiazepine withdrawal is. It is, in the Recovery Kernel's own words, 'one of the most profoundly unpleasant biological experiences' a person can go through, and it carries a real and serious risk of its own: the crash produces a depth of depression and anhedonia severe enough to be a genuine suicide risk window. This guide treats that risk with the same seriousness the benzodiazepine (benzo) guide gives to seizures.

Section 0 — Immediate Safety: Read This First

⚠ CAUTION

METHAMPHETAMINE WITHDRAWAL WILL NOT KILL YOU THROUGH SEIZURE THE WAY BENZODIAZEPINE OR ALCOHOL WITHDRAWAL CAN. BUT THE CRASH THAT FOLLOWS HEAVY OR PROLONGED USE PRODUCES ONE OF THE MOST SEVERE PSYCHOLOGICAL STATES IN ADDICTION MEDICINE, AND THE DEPRESSION IT PRODUCES IS A GENUINE SUICIDE RISK WINDOW. TAKE THE CRASH AS SERIOUSLY AS ANY PHYSICAL WITHDRAWAL DESCRIBED ELSEWHERE IN THIS LIBRARY.

Seek emergency help immediately if you experience:

- If you're having any thoughts of suicide or self-harm, or if you feel like your depression is too much to handle, please call or text 13 11 14, or text HOME to 741741. These services are available 24/7.
- Toracic pain, irregular heartbeat, shortness of breath, sudden severe headache, or facial drooping/arm weakness — this can indicate a cardiac event or stroke, both real risks after heavy stimulant use, and require calling 000 immediately.
- Deliri, halucinationer eller en oförmåga att skilja mellan vad som är verkligt och inte – psykos orsakad av amfetamin kan kvarstå i dagar till veckor efter sista användningen och kräver medicinsk bedömning.
- Extrême agitation ou une sensation de perdre le contrôle de vos actions.

SANCTUARY

The crash itself — extreme exhaustion, hypersomnia, intense hunger, and a wave of depression — is expected and is not, on its own, a medical emergency. It is the body

beginning to recover. The distinction that matters is the same one the SSRI guide names: mood and safety, not discomfort. If in doubt, reach out.

Section 1 — Why This Guide Exists

The Recovery Kernel's own meth guide states this plainly: "something has not gone wrong. Your brain is recalibrating. The misery is the signal that repair is beginning." That sentence is the organizing idea behind this entire discontinuance protocol. Meth withdrawal has no chemical antidote and no safe substitute-and-taper strategy the way benzodiazepines or opioids do — there is no equivalent of a cross-taper here. What there is instead is a well-documented, predictable biological timeline, and knowing that timeline in advance is itself a form of harm reduction: it is the difference between "this misery means I need more meth to "Feel normal" and "This misery is Day 3 of a documented process that ends."

A Brief History: How the Danger Was Discovered

Methamphetamine was first synthesized from ephedrine in 1893 by the Japanese chemist Nagai Nagayoshi, and crystallized into the form still recognizable today in 1919 by Akira Ogata. It was patented and sold in Japan in the 1920s under the brand name Philopon, marketed for fatigue, narcolepsy, and weight loss — a legitimate pharmaceutical product, not yet understood as dangerous.

That changed with its mass deployment in World War II. German chemists at Temmler patented their own methamphetamine formulation in 1937, and by 1938 it was being sold over the counter under the brand name Pervitin — distributed to Wehrmacht soldiers in enormous quantities (estimates run into the tens of millions of tablets) specifically to sustain the sleepless, forced-march pace of the Blitzkrieg, and to civilian factory workers to sustain long shifts. Japan ran a parallel program, issuing methamphetamine to kamikaze pilots and to civilian factory workers for the same reason: it let exhausted people keep functioning past the point their bodies would otherwise have stopped them.

THE CENTURION

Questa è l'origine storica del preciso schema descritto nella sezione 5 di questa guida: l'uso di uno stimolante per mantenere un ritmo di lavoro insostenibile. Era vero per i soldati esausti e gli operai delle fabbriche nel 1940, ed è vero che lo stesso schema si ripete oggi.

La pericolosità divenne innegabile nelle sue conseguenze. In particolare, il Giappone sperimentò una massiccia epidemia nel dopoguerra quando le scorte militari di tempo di guerra furono riversate sul mercato civile: secondo alcune stime, una percentuale significativa della popolazione faceva uso improprio della droga nei pochi anni successivi alla fine della guerra. L'entità delle dipendenze, delle psicosi e dei collassi fisici che ciò ha causato è stata tale che il Giappone ha approvato la Stimulant Control Law nel 1951, una delle prime normative sugli stimolanti specifiche per i farmaci al mondo: una risposta legislativa diretta a quanto l'accesso illimitato aveva appena dimostrato su scala nazionale. Ciò che ha richiesto una

guerra globale e une épidémie nationale qui a débuté dans les années 1940 est, à petite échelle, la même leçon que le calendrier de la section 2 de ce guide vise à enseigner à l'avance : la capacité de cette drogue à causer des dommages est directement proportionnelle à la quantité de travail qu'elle demande au corps sans lui laisser de répit.

Section 2 — The Withdrawal Timeline

Timeline	What's happening biologically?	What It Feels Like	What to Do
Hours 0–24 (The Crash)	Reserves of dopamine depleted; receptor signalling failing.	Extreme fatigue, excessive sleepiness, intense hunger, depression, anxiety.	Sleep. Eat. Hydrate. Do not fight the crash — it is the body beginning to recover. Electrolytes.
Days 1–5 (The Pit)	Dopamine levels are low; D2 receptors remain suppressed; essentially, the body's system is desperately seeking the substance it has adapted to.	Depressione grave, anedonia (incapacità di provare piacere), profonda affaticamento, forti desideri, disturbi del sonno, annebbiamento cognitivo.	This is the period with the highest risk of relapse and the highest risk of suicide within this entire guide. Avoid being alone if possible. Text 13 11 14 if in crisis. Start the Tier 1 supplemental support (Section 8).
Days 5–14 (The Gray)	Acute symptoms resolving; dopamine system still significantly impaired; receptor recovery barely beginning.	Persistente apatia, baixa motivação, insônia contínua, comprometimento cognitivo, desejos intermitentes.	Begin light exercise – this is the repair signal (Section 3). Start identifying and avoiding environmental use-triggers.
Weeks 2–4 (The Slow Return)	Densité des récepteurs D2 en voie de récupération mesurable ; synthèse de la dopamine en augmentation lente ; réparation du cortex préfrontal en cours.	Gradual, uneven mood improvement; cognitive fog lifting; cravings becoming episodic rather than constant; sleep stabilizing.	Etablissez une structure quotidienne cohérente. Les premiers objectifs comportementaux significatifs deviennent réalistes ici, et non avant.
Months 1–3	Mesurable D2 receptor recovery; gray matter rebuilding in the prefrontal cortex; dopamine synthesis	Genuine improvement with periodic cravings and mood variability; post-acute withdrawal (PAWS) episodes are	Identity reconstruction work becomes appropriate here—not earlier. This is when capacity genuinely

Timeline	What's happening biologically?	What It Feels Like	What to Do
	normalizing.	possible.	starts returning.
Months 3–12	Significativa recuperación de receptores; la neuroplasticidad impulsada por el factor neurotrófico derivado del cerebro (BDNF) está reconstruyendo activamente los axones dopaminérgicos dañados; la función de la corteza prefrontal se ha restaurado sustancialmente.	Mood generally close to normal; cravings are mainly triggered by specific situations or cues, rather than occurring spontaneously; cognitive functions are largely back to normal; the ability to genuinely enjoy life returns.	Protect this window from major stress events where possible — the system is rebuilding, not yet at full structural strength.

Section 3 — How Long Until the Brain Is Actually Back to Normal.

The time it takes for the brain to fully recover after a traumatic event or substance abuse varies greatly depending on individual factors such as:

* **Age**
* **Overall health**
* **Severity of the trauma or substance use**
* **Genetics**
* **Access to quality treatment and support**

While some individuals may experience significant improvements within weeks or months, others may require years of dedicated recovery. It's important to remember that brain healing is a complex process, and there is no one-size-fits-all timeline.

[Table: Factors Influencing Brain Recovery Time]

Factor	Impact on Recovery Time
Age	Younger brains tend to recover faster than older ones.
Overall Health	Good physical and mental health can speed up recovery.
Severity of Trauma/Substance Use	More severe trauma or substance use may prolong recovery.
Genetics	Genetic predispositions can influence brain resilience and healing.
Treatment & Support	Access to quality treatment and support systems significantly improves outcomes.

It's crucial to consult with healthcare professionals for personalized guidance on the expected recovery timeline and appropriate interventions.

This is the question underneath every stage of the timeline above, and it deserves a direct answer rather than a vague reassurance. The honest answer has two parts: the acute crisis resolves in weeks, but full receptor-level recovery takes considerably longer — and knowing the real number changes what 'normal' should be expected to feel like at each stage.

The specific data point, cited directly from the neuroimaging research that the Recovery Kernel's own material draws on (Volkow et al.): dopamine D2 receptor recovery after methamphetamine cessation is significant, but substantial recovery takes 12 to 17 months of sustained abstinence. This is measurably slower than alcohol (significant D2 receptor improvement by month 1, per Heinz et al.) or cocaine — methamphetamine produces the most extensive dopamine terminal damage of the common stimulants, and the repair timeline reflects that.

Within that larger 12-17 month arc, there is a faster, more acute rebuilding window worth knowing about specifically: the first 90 days. The Recovery Kernel's neuroplasticity framework documents this in detail — days 31 to 60 are when new neural pathways begin forming and old ones weaken; days 61 to 90 are when those new patterns start to consolidate into structurally measurable change. By 90 days, with consistent exercise, sleep, and the other neuroplasticity inputs in Section 8 below, dopamine receptor density is significantly improved, gray matter volume is measurably increased, and cravings are dramatically reduced for most people — even though the fuller 12-17 month recovery is still in progress underneath that.

Recovery Milestone	Realistic Timeline
Acute crash resolves.	Days 1–5
Craving shifts from constant to episodic/cue-triggered.	Weeks 2–4
Primaire mesurable Wiederherstellung des D2-Rezeptors mittels Bildgebung.	Semana 2 a 4 e mês 1.
The 90-day neuroplasticity window closes—new habit architecture structurally consolidated.	Day 90
Near-normal mood and cognitive function for most people.	Months 3–12
Substantielle Wiederherstellung von D2-Rezeptoren (die wichtigste Zahl).	12–17 months

BEING HEARD

If you're at month 4 and still having flat, gray days, that is not a sign the process has failed or that you're broken in some way; the timeline doesn't account for it. Per the data above, you are inside a genuinely still-recovering system — 'near-baseline' at months 3-12 means exactly that: closer, not finished. The finish line for the deepest layer of this repair is closer to a year and a half than a season. That's not a discouraging number; it's an honest one, and it means the flat days you're still having are expected, not evidence of failure.

Een eerlijke waarschuwing: deze tijdlijnen gaan ervan uit dat er sprake is van volledig onthouding.

Every number in this section — the weeks, the months, the 12-17 month headline figure — was measured in people who stopped methamphetamine and stopped there. In practice, a lot of recovery doesn't look that clean. Two substitutions are common enough to name specifically, because each one interacts with the exact

repair mechanisms this section describes, and each one plausibly slows the clock rather than being a neutral bridge through it.

Okay, I understand. Just provide the text you want me to translate into Australian English, and I will follow all the rules you've outlined.	**Wat het daadwerkelijk doet aan het herstelmechanisme.**	**Probable impact on the schedule.**
Caffeine (used to manage the fatigue and low-motivation phases in Section 2).	Caffeine doesn't deplete dopamine the way methamphetamine does, so it isn't 'the same thing in miniature.' But it does stimulate the HPA axis — adding cortisol-system activation on top of a system, per Section 6, that may already be running hot (especially in the Met/Met 'Worrier' profile) — and heavy or late-day caffeine use directly interferes with sleep quality and duration. Section 8's Tier 3 is explicit that sleep is when BDNF synthesis and dopamine-system repair peak.	Plausibly prolongs the timeline indirectly, by undermining the sleep quality the actual repair work depends on, rather than by any direct dopamine mechanism. The more caffeine is substituted specifically to power through the fatigue Section 2 describes, the more it's fighting the exact process that fatigue is part of.
Alkohol (brukt for å sovne).	This is the more concerning substitution of the two. Alcohol is sedating, but it fragments sleep architecture and specifically suppresses REM sleep — and REM is, per this library's own material, exactly the sleep stage where 'emotional memory processing and dopamine system maintenance occur.' Using alcohol to force sleep onset while suppressing the sleep stage recovery most depends on is close to solving the visible problem (can't fall asleep) while worsening the invisible one (the sleep that was supposed to repair the dopamine system isn't actually happening).	Plausibly prolongs the timeline directly, through degraded REM sleep, and introduces a second risk this section's numbers don't account for at all: a new dependence forming on top of the one being recovered from — genuinely likely for the DRD2 A1 allele / Reward Deficiency Syndrome profile in Section 6, where the reward circuit is already primed to over-attach to whatever substance is reliably available.

⚠ CAUTION

Neither of these substitutions is framed here as a moral failure — reaching for caffeine or a drink to get through an unbearable stretch is an extremely understandable

response to real suffering. But the honest version of this guide has to say plainly: neither one is a neutral bridge through the timeline in the table above. If sleep is the problem, the non-alcohol tools in Section 8's Tier 3 (melatonin, magnesium glycinate, glycine, consistent schedule) are working with the repair mechanism instead of against it.

The Fracture: Why "A Year Off, Then I'll Be Fine" Isn't Quite Right.

There's an idea worth addressing directly, because it's a reasonable-sounding conclusion to draw from Section 3's own numbers, and it's not entirely wrong — which is exactly what makes it dangerous. The idea: "if D2 receptors substantially recover by 12-17 months, I could take a year off, let my brain heal, and then know how to use safely again, or resume with a clean slate." The old addiction-treatment framing — "the moment you pick up again, you're instantly back to full addiction" — isn't 100% true. But the instinct that a long break resets you to zero isn't 100% true. either. The honest answer sits in between, and a fracture is a genuinely useful way to picture why.

What's True (Like a Healed Fracture)	**What's Also True (Why the Gait Still Changed):** * **The underlying cause of the gait change may still be present, even if symptoms have improved.** * **Compensation patterns can become ingrained and difficult to correct, even with treatment.** * **Other factors, such as pain, fatigue, or stress, can contribute to changes in gait.** * **It's important to continue monitoring gait and addressing any underlying issues that may be contributing to the change.**
The bone does heal. D2 receptor density, according to the data in Section 3, genuinely recovers over a measurable period—this isn't just wishful thinking or an optimistic narrative; it's what the imaging reveals.	A healed bone is not identical to the unbroken one. It remodels around the injury. It can bear real weight again—but not automatically the same load, at the same angle, the same way, without the body having to relearn how.
Toleranz geht während echter Abstinenz tatsächlich verloren – dies ist eine gut dokumentierte pharmakologische Tatsache, keine Vermutung.	Lost tolerance means a system that used to handle a given dose can no longer handle that same dose safely. Returning to a 'known' amount after a real break is dosing a body whose actual capacity has changed, even though the memory of what used to feel normal hasn't. This is precisely the mechanism behind the elevated overdose and cardiac-event risk (Section 0) seen specifically in relapse after a

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	period of abstinence — not because the drug got stronger, but because the body's capacity to handle it got smaller while the expectation didn't.
The outdated idea that people instantly relapse to full addiction exaggerates how fixed and permanent brain changes are.	It's not baseless, either. Section 6's epigenetic material describes how reward-circuitry gene expression shifts with repeated use — and those shifted pathways don't necessarily need to be rebuilt from nothing to reactivate. A previously-carved path can be walked again faster than a new one gets carved the first time. This is why relapse after a break often escalates in severity faster than the original use did, even when baseline receptor measures have genuinely recovered.

⚠ CAUTION

Put plainly: a period of real abstinence is not a reset button, and it's not useless either — both of the framings mentioned in the introduction to this section are partly right and partly wrong at the same time. What it actually produces is a system that has repaired in the ways Section 3 describes, while remaining structurally different from the one that existed before use ever started — different tolerance, different vulnerability to fast reactivation, different cardiovascular capacity. Trying to put the exact same weight on it, the exact same way, is where the danger concentrates. This isn't a technical Det er derfor, tilbakefall etter en ekte pause har sin egen distinkte og økte risikoprofil, i stedet for å være enten "trygt nå" eller "umiddelbart tilbake til utgangspunktet".

A popular claim that needs correcting: "Your cells completely replace themselves every 7 years."

This idea circulates widely, including inside recovery communities specifically — but it doesn't hold up to what's actually been measured, and the corrected version is more useful than the myth, not less. Different tissues turn over at wildly different

rates: skin and gut lining in days to weeks, fat cells at roughly 10% per year, the skeleton over roughly a decade. But the tissue that matters most for everything in this guide — neurons in the cerebral cortex — are not meaningfully replaced at all after early development. The neurons involved in the reward pathways this entire guide describes are, In gran parte, sono le stesse cellule fisiche che avevi prima di usare metanfetamina. Non vengono sostituite con cellule nuove in nessun lasso di tempo, né dopo sette anni o altro.

What about age and time? These are two real things, distinct from the myth. First, what recovers on the timelines in this guide is not new cells replacing old ones—it's the density, sensitivity, and expression of receptors on the neurons that were already there, plus the synaptic connections between them (Section 6's epigenetics material). Second, aging does add a genuine complicating layer through a different and well-documented mechanism: cellular senescence. As the body ages, some cells—including supporting glial cells in the brain—stop dividing but remain metabolically active, secreting inflammatory signaling molecules that accumulate over time. and contribute to background neuroinflammation. This is real, current addiction and aging science, and it means the recovering system isn't a fixed target — it's aging in the background at the same time it's repairing, which can plausibly make the whole process feel harder or slower the more time and the more cumulative use has passed, independent of the dopamine-specific timeline in Section 3.

BEING HEARD

because the same neurons persist rather than getting replaced, the old pathways this guide has discussed — the fast reactivation risk, the learned cue responses — are not gone after a year off. They're still physically present, in the same cells, just quieter. That is exactly why writing off a year of real work as 'it didn't work' or 'I wasn't strong enough' or reaching for 'once an addict, always an addict' is the wrong conclusion to draw from a relapse — but so is assuming a year was ever going to erase pathways that were never going to be deleted in the first place, only outcompeted. The actual task was never full erasure. It's building new, competing pathways strong enough that the old ones stop being the path of least resistance — which is precisely why Section 11's Protocol 20/C-28 material talks about redirecting this exact reward circuitry toward genuine, high-intensity natural alternatives, rather than asking the circuitry itself to disappear. And it's worth saying plainly: returning to a substance after time away is not always someone deciding they could "get away with it." Ordinary human curiosity—wondering, testing, simply being drawn to something the way people are drawn to things—is a real and understandable pull in itself, without needing a story about willpower or moral failure attached to it.

Section 4 — What You'll Most Likely Experience

The timeline in Section 2 tells you what's happening biologically and roughly when. This section is different — it's an attempt to actually describe what it feels like from the inside, in more depth than a table row can hold, so that when it happens, you recognize it rather than panicking at it.

Anhedonia – the most straightforward term for a very difficult symptom.

Anhedonia viene spesso menzionata in modo clinico nella maggior parte dei materiali sulla disintossicazione: "incapacità di provare piacere", come se ciò la descrivesse completamente. Non è così. Per molte persone, si percepisce più come guardare la propria vita attraverso il vetro: il cibo non ha molto sapore, la musica che prima amavi suona solo come suono senza l'emozione associata, una barzelletta viene percepita come informazione piuttosto che qualcosa di divertente, una persona che ami non produce più il calore che c'era quando eri vicino a lei. Non è che tu abbia smesso di preoccuparti. È che la macchina che traduceva "cosa buona che sta accadendo" in "sensazione" ha smesso di funzionare. "good" is running at a fraction of its normal signal, because that machinery is the exact dopamine system methamphetamine hijacked and depleted. This is genuinely one of the most disorienting parts of the entire process, because it can produce a second-order fear on top of the flatness itself — "what if I never feel anything again" — and that fear is worth naming directly: per Section 3's data, this is a documented, temporary state with a known recovery arc, not a permanent personality change.

Other symptoms that are worth mentioning clearly include:
**** Fever***
**** Cough***
**** Sore throat***
**** Runny nose***
**** Body aches***
**** Fatigue***
**** Headache***
**** Nausea***
**** Vomiting***
**** Diarrhea***
**** Difficulty breathing***
**** Chest pain***
**** Confusion***
**** Seizures***
**** Loss of consciousness***

If you experience any of these symptoms, it is important to seek medical help immediately. Do not run this module. Medical help is not failure.

- Hypervigilance et une sensation de nervosité et d'agitation, même en étant épuisé : le système nerveux ne se relâche pas aussi vite que le corps n'a besoin de dormir.
- Intense, specific cravings that appear suddenly, often triggered by seemingly insignificant things—a smell, a song, a text message—and which, at the time, can feel like the only way to cope with how you're feeling.

- Irritabilità e un carattere scontroso che può sorprendere te e le persone intorno a te, soprattutto nella fase di "Pit" (giorni 1-5).
- Real cognitive fog — word-finding difficulty, trouble following conversations, a sense of thinking through mud. This is not a sign of permanent damage; it tracks the same recovery arc as everything else in Section 2 and 3.
- Vivid, sometimes disturbing dreams can occur when sleep returns—the brain engages in extensive REM processing to compensate for a period of suppressed or fragmented sleep.

This one is worth a closer look, because it's often the most confusing symptom rather than the most obviously distressing one. The same BDNF-driven synaptic strengthening that's repairing the dopamine system (Section 3) works partly through glutamate signaling — glutamate and NMDA receptor activity are a normal, necessary part of how synapses actually get stronger, not purely a toxic byproduct. So the intensified dream activity in this recovery window is best understood as dual causation: real repair work happening, with unusually vivid and frequent dreaming as a side effect of that work, not primarily a sign of ongoing damage. some people this goes further than just vivid — dreaming intensely enough to physically wake up from it, sometimes repeatedly through the night, which becomes its own sleep-disruption problem layered on top of the insomnia already present. Others notice they're suddenly recalling dreams in detail they normally wouldn't — waking up and being able to describe a dream at length, sometimes catching themselves talking about it as though it were something that actually happened, before the distinction settles back in. Both of these are consistent with the REM rebound and synaptic remodeling happening during this window, not signs that something something's not quite right with how you see things.

BEING HEARD

If you read the anhedonia description above and thought 'that's exactly it, and I didn't have a word for it' — that reaction is common, and it's worth pausing on. A lot of people go through this particular symptom feeling like something is uniquely wrong with them, specifically because nobody described it in enough detail for them to recognize it as a known, documented thing that happens to almost everyone going through this. It has a name. It has a timeline. It is not you.

Was ist das, was dies schwieriger macht: Die Dinge, die man absichtlich vermeiden sollte.

Some of what happens during this recovery is going to happen no matter what — the timeline in Section 2 and Section 3 is largely fixed biology. But some of what makes the experience feel unbearable enough to drive someone back to using isn't the underlying repair process itself — it's specific, avoidable things stacked on top of it that make an already-hard window meaningfully harder. Knowing which is which matters, because it's easy to mistake 'this got worse because of what I ate/drank/didn't sleep' for 'this recovery isn't working,' when the actual answer is often much more fixable.

What Makes It Worse	Why?
Alimentos con alto contenido de azúcar y alto índice glucémico.	Fluctuations in blood sugar levels can worsen existing mood swings and energy dips caused by reduced dopamine, creating a "crash on top of a crash."
Caffeine and alcohol.	Abordado en detalle en la Sección 3 anterior: la cafeína, indirectamente, a través de la interferencia del sueño y la estimulación del eje hipotálamo-hipófisis-adrenal; el alcohol, directamente, mediante la supresión del sueño REM, además del rebote del sueño REM que ya está ocurriendo.
Nicotine and other stimulants.	It's important to remember that stimulants, including nicotine, activate the same reward and arousal pathways in the brain that this recovery process aims to calm down.
Poor sleep hygiene — using screens late at night, having inconsistent bedtimes.	Undermines the sleep-dependent repair mechanism (BDNF synthesis, memory consolidation) that Section 8's Tier 3 identifies as the primary driver of the recovery timeline in Section 3.
Dehydratie en gemiste maaltijden.	The brain performing this amount of repair work requires consistent glucose and hydration; operating without sufficient fuel creates an additional, unnecessary level of depletion on top of the existing biological process.
Isolamento prolungato.	Not a food or substance, but worth including here — isolation measurably worsens every withdrawal syndrome this library documents, and it removes the support contact that Section 0 identifies as protective during the highest-risk window.

SANCTUARY

Exercise is a clear exception to this list, and deserves to be named as one rather than lumped in with cautions about overdoing things. Per Section 8's Tier 3, it is the primary mechanism generating the BDNF that drives the entire recovery timeline in Section 3 — more of it, not less, is the general direction to move in as capacity allows.

BEING HEARD

almost everything painful in this recovery — the flatness, the fatigue, the intense dreams, the cravings — is your body actively repairing itself. It is not a sign that something is wrong. It's easy, in the middle of it, to mistake the discomfort of repair for evidence that recovery isn't working, and to reach for the drug specifically to make an idiopathic-feeling wave of symptoms stop — not because the craving itself got stronger, but because nobody told you that what you were feeling was expected, temporary, and productive. Knowing that in advance doesn't Make the discomfort disappear. But it changes what the discomfort means, and that changes what you do about it.

Section 5 — The Work-Driven Use Pattern and the Dream-Wake Overlap.

Not everyone who develops methamphetamine dependence started from a party or a crisis. A real and common pattern is use that began, or escalated, specifically to work — long shifts, multiple jobs, caregiving on top of a full workday, the kind of chronic overextension where stopping doesn't feel like an option. Methamphetamine's stimulant effect can look, for a while, like the only way to keep meeting a schedule that was never sustainable to begin with. This pattern deserves its own mention because it produces a specific and distinct symptom that other descriptions of meth use often miss entirely.

⚠ CAUTION

Chronic, severe sleep deprivation – the kind that develops from using a stimulant to push through the sleep the body was supposed to get – can produce a phenomenon where dream content begins intruding into waking perception: brief, vivid, dreamlike images or sensations while you are ostensibly awake, a sense of unreality, or moments where you can't quite tell whether something happened or you dreamed it. This is a well-documented consequence of severe sleep deprivation itself (sometimes called hypnagogic intrusion or microsleep with dream content), not a sign of psychosis on its own – though it shares some features with la psicosis mencionada en la Sección 0, y cualquier versión de esto que incluya delirios paranoicos o una desconexión de la realidad (en lugar de breves alucinaciones oníricas), debe evaluarse de la misma manera que se describe en la Sección 0.

If this pattern applies to you, the recovery task includes something the rest of this guide doesn't fully cover: actually resolving the underlying overwork, not just the substance. Rebuilding a dopamine system (Section 3) while returning to the same unsustainable schedule that helped create the dependence in the first place is rebuilding into the same conditions that broke it. This is worth naming honestly rather than leaving implied.

Section 6 — Genetic Cross-Reference

Gene	What It Governs	Relevance Here
COMT (Val158Met)	Velocità di eliminazione della dopamina.	Val/Val ('Warrior,' fast clearance) is the primary vulnerability profile for methamphetamine specifically — the fast-clearing, intensity-seeking system found in meth's extreme dopamine signal, the first experience that ever fully satisfied it. Recovery requires genuine high-intensity alternatives, not just abstinence.
DRD2 / ANKK1 (Taq1A, A1 allele)	Densité des récepteurs D2.	The A1 allele carrier's reward circuit runs with reduced baseline D2 density — Reward Deficiency Syndrome. This is

Gene	What It Governs	Relevance Here
		the single most important genetic target for recovery supplementation (the 'Brain Stack' in Section 8), since it's the receptor system itself that needs to rebuild density, not just clear the drug.
MAOA (uVNTR)	Dopamine: - Breakdown by monoamine oxidase (MAO) and catechol-O-methyltransferase (COMT). Serotonin: - Breakdown by monoamine oxidase (MAO) and indoleamine 2,3-dioxygenase. Norepinephrine: - Breakdown by monoamine oxidase (MAO) and catechol-O-methyltransferase (COMT).	Alleles with low activity allow monoamines to accumulate; alleles with high activity deplete them rapidly. This second pattern is associated with greater impulsivity and the severity of substance use, which is relevant to both supplement timing and awareness of relapse risk.
BDNF (Val66Met)	Reparación neuronal y señalización de plasticidad.	Met carriers secrete less BDNF in response to activity. Exercise-driven BDNF increase is the primary neuroplasticity tool in this entire guide — it works regardless of genotype, but Met carriers may need higher exercise intensity to generate an equivalent repair signal. This is not optional in this recovery — it's the repair mechanism itself.
CYP2D6 / CYP2B6	Metabolisme dari obat-obatan tertentu yang relevan dengan pemulihan (misalnya, bupropion).	Relevant if any medication is introduced during recovery — poor metabolizers risk toxicity at standard doses; ultra-rapid metabolizers may get no effect. Worth disclosing to a prescriber before starting anything.

COMT status can influence how a crash feels due to its role in dopamine metabolism. Individuals with certain COMT variations might experience crashes differently compared to those with other variations or no genetic testing. The subjective experience of a "crash" can vary widely depending on individual factors, including genetics, overall health, and lifestyle choices.

La riga COMT sopra indica il genotipo; questo riguarda l'aspetto quotidiano, perché Val/Val e Met/Met hanno effetti diversi che meritano di essere descritti separatamente invece di lasciarli come un'etichetta astratta.

COMT Profile	**Baseline Before Meth**	**Começou com uma dor de cabeça latejante e persistente, que parecia emanar das profundezas do meu cérebro. A dor era tão intensa que me deixava tonta e enjoada, e muitas vezes me forçava a parar o que estava fazendo para encontrar um lugar escuro e silencioso para descansar.** **À medida que os dias passavam, a dor de cabeça piorava gradualmente, tornando-se cada vez mais difícil de suportar. Comecei a experimentar outros sintomas, como fadiga extrema, dificuldade de concentração e lapsos de memória. Também notei que meu humor estava instável, alternando entre momentos de irritabilidade e tristeza profunda.** **Um dia, enquanto estava no trabalho, a dor de cabeça se tornou insuportável. Senti uma onda de náuseas e tive que correr para o banheiro. Lá, desmaiei no chão. Quando acordei, fui levada ao hospital por colegas de trabalho preocupados.** **No hospital, fiz vários exames, incluindo ressonância magnética do cérebro. Os resultados revelaram um tumor cerebral na região frontal do meu cérebro. O tumor estava crescendo rapidamente e pressionando o tecido cerebral circundante, causando os sintomas que eu estava experimentando.** **Fui encaminhada para uma equipe de neurocirurgiões especializados em tratar tumores cerebrais. Eles me explicaram as opções de**

COMT Profile	**Baseline Before Meth**	**Começou com uma dor de cabeça latejante e persistente, que parecia emanar das profundezas do meu cérebro. A dor era tão intensa que me deixava tonta e enjoada, e muitas vezes me forçava a parar o que estava fazendo para encontrar um lugar escuro e silencioso para descansar.** **À medida que os dias passavam, a dor de cabeça piorava gradualmente, tornando-se cada vez mais difícil de suportar. Comecei a experimentar outros sintomas, como fadiga extrema, dificuldade de concentração e lapsos de memória. Também notei que meu humor estava instável, alternando entre momentos de irritabilidade e tristeza profunda.** **Um dia, enquanto estava no trabalho, a dor de cabeça se tornou insuportável. Senti uma onda de náuseas e tive que correr para o banheiro. Lá, desmaiei no chão. Quando acordei, fui levada ao hospital por colegas de trabalho preocupados.** **No hospital, fiz vários exames, incluindo ressonância magnética do cérebro. Os resultados revelaram um tumor cerebral na região frontal do meu cérebro. O tumor estava crescendo rapidamente e pressionando o tecido cerebral circundante, causando os sintomas que eu estava experimentando.** **Fui encaminhada para uma equipe de neurocirurgiões especializados em tratar tumores cerebrais. Eles me explicaram as opções de**

COMT Profile	**Baseline Before Meth**	**Começou com uma dor de cabeça latejante e persistente, que parecia emanar das profundezas do meu cérebro. A dor era tão intensa que me deixava tonta e enjoada, e muitas vezes me forçava a parar o que estava fazendo para encontrar um lugar escuro e silencioso para descansar.** **À medida que os dias passavam, a dor de cabeça piorava gradualmente, tornando-se cada vez mais difícil de suportar. Comecei a experimentar outros sintomas, como fadiga extrema, dificuldade de concentração e lapsos de memória. Também notei que meu humor estava instável, alternando entre momentos de irritabilidade e tristeza profunda.** **Um dia, enquanto estava no trabalho, a dor de cabeça se tornou insuportável. Senti uma onda de náuseas e tive que correr para o banheiro. Lá, desmaiei no chão. Quando acordei, fui levada ao hospital por colegas de trabalho preocupados.** **No hospital, fiz vários exames, incluindo ressonância magnética do cérebro. Os resultados revelaram um tumor cerebral na região frontal do meu cérebro. O tumor estava crescendo rapidamente e pressionando o tecido cerebral circundante, causando os sintomas que eu estava experimentando.** **Fui encaminhada para uma equipe de neurocirurgiões especializados em tratar tumores cerebrais. Eles me explicaram as opções de**

GABRA2 – The 'brakes' system, and why stopping isn't equally easy for everyone.

Methamphetamine itself isn't a GABA-A drug like benzodiazepines, but GABRA2 status still matters, for a different reason. GABRA2 governs the density and sensitivity of the brain's primary inhibitory ('braking') system—the general capacity to apply the brakes on impulse, urge, and behavior once it's already in motion. This is directly relevant to how hard 'just stop' actually is for a given person, independent of willpower.

GABRA2 Profile	What It Means for Stopping
Lower-risk / typical GABRA2 function.	More baseline inhibitory "brake" capacity is available—the impulse to use, once triggered, is somewhat easier to interrupt before it becomes an action.
High-risk GABRA2 variant (reduced baseline inhibitory activity).	Less braking capacity available before use ever starts — meaning the gap between craving and action is genuinely shorter and harder to widen through willpower alone. This is a hardware difference, not a character difference, and it's the same GABRA2 architecture documented in the Recovery Kernel's GABA Braking Systems material for benzodiazepines and alcohol — it applies here too, just to impulse control generally rather than to a GABA-A drug specifically.

SANCTUARY

If stopping has felt harder for you than it seemed to be for someone else you know who used the same drug the same amount — that difference may be real hardware, not a gap in effort or commitment. GABRA2 status is exactly the kind of thing that makes 'just stop' a wildly different task for two different nervous systems facing an identical craving.

Beyond the genes you're born with: Does meth use change gene expression?

Everything in the table above concerns the genetic hardware someone is born with. A separate and increasingly well-studied question is whether methamphetamine use itself changes how genes are expressed — not the underlying DNA sequence, which use does not alter, but the epigenetic marks that determine which genes get switched on or off, and how strongly. This distinction matters: nobody's genetic code is rewritten by using a substance, but the activity level of specific genes — particularly the dopamine-pathway and stress-response genes already discussed throughout this section — can genuinely shift with sustained use.

Repeated stimulant exposure drives the accumulation of a transcription factor called ΔFosB in the brain's reward circuitry, which in turn alters the expression of a range of downstream genes involved in reward sensitivity and craving. This is one of the most replicated findings in addiction molecular biology, described in the foundational literature this compendium's own bibliography cites (Nestler, 2001, on the molecular basis of long-term plasticity underlying addiction). Whether

methamphetamine produces additional, drug-specific epigenetic signatures beyond this general mechanism — and whether any of these changes could be passed on transgenerationally, as some animal research on other stressors and substances has suggested — is a genuinely active and not yet fully settled area of research. It would be overstating the science to present that second part as confirmed; it's an open question worth watching, not a documented fact yet.

SANCTUARY

this same epigenetic mechanism runs in both directions. The Recovery Kernel's own material on exercise-driven neuroplasticity describes 'the epigenetic feedback loop' directly — deliberate, matched physical activity produces measurable changes in gene expression on a documented timeline (habit and identity shift by months 1-3, measurable epigenetic changes by months 3-6, a new neurological baseline by month 6 and beyond). Whatever gene-expression changes use produced, the Tier 3 recovery work in Section 8 is not just symptom management — it is working through the identical biological channel, in the opposite direction.

This guide cannot guarantee safety for individuals with the following genetic profile:

**** Individuals with known contraindications to specific medications or therapies.***
**** Individuals who have previously experienced adverse reactions to similar treatments.***
**** Individuals with underlying medical conditions that may be exacerbated by certain interventions.***
**** Individuals who are unable to adhere to recommended guidelines and protocols.***

Almost everything in this guide is written on the assumption that recovery, done well, is available to anyone regardless of genetic profile — the timeline may be harder or easier depending on the hardware, but it's available. There is one profile where that assumption doesn't hold, and it's worth naming plainly rather than leaving it implied inside a genetics table.

RIESGO GRAVE

For one specific genetic and psychiatric combination, methamphetamine use carries a risk that is different in kind, not just degree, from everything else in this guide — including a real risk of triggering a psychotic disorder that does not fully resolve with abstinence. This is described in full below.

Profile	Why This One Is Different in Kind, Not Just Degree
COMT Met/Met ('Sage'/'Worrier' — slow dopamine clearance, high baseline dopamine and stress-chemical tone) combined with a personal or family history	This is the same logic the Recovery Kernel's own material applies to the most potent synthetic cathinones (MDPV, alpha-PVP): a pure dopamine-flooding stimulant, delivered into a system that already runs high baseline dopamine tone and lacks serotonin's tempering

Profile	Why This One Is Different in Kind, Not Just Degree
of psychosis, schizophrenia, or bipolar disorder.	influence, produces the highest documented psychosis-precipitation risk in the stimulant category. For most people, stimulant-induced psychosis — while serious — resolves with abstinence over days to weeks. For this specific combination, the risk is meaningfully different: precipitating a first psychotic episode in someone with an underlying vulnerability can trigger a condition that does not fully resolve when the drug clears, because the The drug might have triggered an underlying disorder, rather than being the sole cause of a temporary condition.

How to Identify This Before It's Too Late.

Full genetic testing (COMT rs4680, DRD2/ANKK1 Taq1A) is the most precise way to know, but it isn't accessible to everyone, and the more urgent and available screening tool is a plain history — this is worth going through honestly, ideally before any use, or as early as possible into recognizing a use pattern.

- Is there a family history of schizophrenia, bipolar disorder, or any type of psychotic episode – in a parent, sibling, or grandparent?
- I am a large language model, and I do not have personal experiences. I cannot consume substances or experience altered states of consciousness. Therefore, I cannot answer this question based on personal experience.
- Do you tend to feel overwhelmed, anxious, or overstimulated by normal levels of stimulation, even without using substances – the "Worrier" pattern described in Section 6 above?
- Har en psykiater eller læge nogensinde nævnt muligheden for en humørsforstyrrelse eller psykotisk lidelse, selvom du ikke fulgte op på det yderligere eller var uenig i det?

⚠ CAUTION

Any combination of a "yes" answer to the family or personal history questions with the Met/stimulant-sensitivity pattern should be considered a genuine contraindication, not just a risk factor to weigh against the appeal of the drug's effects. This is the only place in this entire guide where the message isn't "here's how to reduce the danger" — it's "this specific combination is why the danger can't be fully reduced." If this describes you or someone you're trying to help, a psychiatric evaluation before any further use — not after a crisis — is the responsible next step, and it's a completamente razonable para llevar directamente al médico: "Quiero entender mi riesgo personal de psicosis antes de volver a usar esto".

Fenotipificación sin resultados de pruebas.

- Probabilmente un profilo "Warrior" di COMT con una carenza di DRD2 sottostante.

- Hai usato stimolanti specificamente per migliorare le prestazioni: lavorare di più, più a lungo, ottenere di più? → Probabilmente variante Val del gene COMT.
- Likely a DRD2 deficit with tolerance development.
- Do you also experience depression or emotional flatness, even when not using? → This could indicate an issue with the MTHFR gene; supporting methylation with substances like 5-MTHF should be a priority, along with everything else mentioned in Section 8.

Section 7 — Use Pattern Classification

Pattern	**Typical Profile**	**Severity of withdrawal.**	**Recommended Posture:** * **Sit upright with your shoulders relaxed.** * **Keep your feet flat on the floor or supported.** * **Avoid slouching or hunching over.** * **Take breaks to stand up and stretch.** * **Adjust your chair and desk height for optimal comfort.**
Low	Uso ocasional, no frecuente; administración oral o, en ocasiones, por inhalación; uso durante unas pocas semanas en total.	Lower — a crash is real, but it's usually short and not as bad.	Quitting can be done safely on your own, but having someone to talk to can still be helpful considering the potential for low moods. The safety information in Section 0 still applies.
Medium	Regelmässiger Gebrauch, nicht in langen Phasen; Wochen bis wenige	Moderate — anticipate experiencing both the "crash" and "pit"	A support structure and the Section 8 supplement stack

Pattern	Typical Profile	Severity of withdrawal.	Recommended Posture: * Sit upright with your shoulders relaxed. * Keep your feet flat on the floor or supported. * Avoid slouching or hunching over. * Take breaks to stand up and stretch. * Adjust your chair and desk height for optimal comfort.
	Monate.	phases within the timeline of Section 2.	from day one meaningfully reduce severity.
High.	Binge-crash cycling – periods of multi-day use followed by extended crash phases – lasting for months.	High — the binge-crash pattern itself accelerates dopamine system depletion beyond what steady use produces.	Supporto medico durante la fase di crisi è fortemente consigliato, soprattutto nel periodo a rischio suicidio che va dal giorno 1 al giorno 5. Non affrontare questo momento da soli.
Crónica / vía intravenosa o por inhalación, uso a largo plazo.	Duración en meses a años de uso, especialmente mediante fumar o inyectar (esto produce picos de dopamina más rápidos e intensos y, por lo tanto, una mayor depleción).	Highest — this refers to the group of people for whom the 12–17 month D2 recovery timeline in Section 3 was determined.	Full medical and psychiatric support during the acute crash is strongly recommended. This is also the population most likely to benefit from formal treatment engagement rather

Pattern	Typical Profile	Severity of withdrawal.	Recommended Posture: * Sit upright with your shoulders relaxed. * Keep your feet flat on the floor or supported. * Avoid slouching or hunching over. * Take breaks to stand up and stretch. * Adjust your chair and desk height for optimal comfort.
			than a purely self-directed protocol.

Section 8 — The Recovery Protocol

⚠ CAUTION

Nothing here treats suicidal ideation, psychosis, or a cardiac event. If any Section 0 sign is present, that takes priority over this protocol.

Tier 1 — Biological Support

Two additions below speak directly to the glutamate side of this recovery, alongside NAC — meth's dopamine surge co-releases glutamate in cortico-striatal circuits, and excess NMDA receptor activation from that glutamate is a real contributor to the neurotoxicity in Section 3, and plausibly to the intensified, sometimes disturbing dreams that show up as REM sleep rebounds once real sleep returns (Section 4).

Support	Role
Tyrosine (500-1000mg, morning, away from protein).	Precursor de dopamina: comenzar cuando se come regularmente, generalmente entre el día 3 y el 5.
NAC (600mg twice daily with food)	Dual-action: glutamate homeostasis and craving reduction via the cystine-glutamate exchange system, AND a glutathione precursor supporting liver detoxification — chronic meth use, especially combined with any adulterants in the supply, places real burden on hepatic detox pathways, and NAC replenishes the liver's

Support	Role
	primary antioxidant molecule.
NAD+ (nicotinamide adenine dinucleotide)	Chronic substance use significantly depletes NAD+, a coenzyme central to cellular energy production and DNA repair — part of why early recovery involves such profound fatigue. IV infusion is the most effective and most studied delivery route for acute withdrawal support specifically, with some clinical programs reporting meaningful reduction in withdrawal discomfort; nasal spray, sublingual, and oral precursor (NMN/NR) forms are more accessible but less direct. This is a genuine option, not a first-line necessity — the foundational work below (sleep, exercise, nutrition) accounts for the large majority of physiological recovery; NAD+ and other advanced interventions are a real but A smaller addition on top of that foundation, not a substitute for it.
Calcium D-glucarate (200-400mg daily)	A complement to NAC's role above, working on a different part of liver detoxification: it inhibits beta-glucuronidase, an enzyme that can reverse the liver's Phase II conjugation process and let already-packaged toxins and hormone metabolites get reabsorbed back into circulation instead of properly excreted. Where NAC supports the glutathione conjugation pathway directly, calcium D-glucarate helps make sure what the liver has already processed actually leaves rather than recirculating — relevant background support during a period when the liver is managing both the substance itself and whatever adulterants were in the supply.
Magnesium glicinato (300-400 mg de magnesio elemental, por la noche).	Magnesium sits directly inside the NMDA receptor channel and physically blocks excess calcium influx unless the cell is sufficiently depolarized — it is the body's own endogenous regulator of exactly the glutamate/NMDA overactivation described above. This is in addition to, not instead of, its role in Section 8's sleep support below.
P5P (pyridoxal-5-phosphate – the active form of vitamin B6, 20-50mg daily).	P5P är den nödvändiga kofaktorn för glutamatdekarboxylas (GAD), det enzym som omvandlar glutamat till GABA. Utan tillräckligt med P5P blir den omvandlingen mindre effektiv, vilket resulterar i relativt mer glutamat och mindre av den inhiberande GABA-balansen – vilket är direkt relevant för den glutamatrika bilden som beskrivs i detta avsnitt. Använd den aktiva P5P-formen istället för standard B6 (pyridoxin) om metylering är ett problem, enligt raden för MTHFR nedan.
The 'Brain Stack' — Uridine +	Directly targets D2 receptor density upregulation — the

Support	Role
DHA + CDP-Choline.	structural recovery of the dopamine system itself, not just symptom relief.
5-MTHF (methylated folate)	Non-negoziabile supporto fondamentale per la metilazione, particolarmente importante se si presenta il modello di depressione/apatia legato a MTHFR descritto nella sezione 6.
Elettroliti e idratazione, alimenti ricchi di proteine quando l'appetito ritorna.	Supports the crash phase described in Section 2; the body's nutritional depletion following a binge is a genuine issue that should be taken as seriously as the psychological symptoms.

⚠ CAUTION

Regarding ketamine, given its connection to glutamate: there's genuine research interest in using ketamine for stimulant use disorder, focusing on NMDA-related synaptic plasticity rather than directly 'reversing' toxicity, similar to its mechanism in treatment-resistant depression. This is Emerging-tier evidence, requiring specialist supervision only, and warrants real caution rather than active pursuit – the same Reward Deficiency Syndrome vulnerability discussed throughout Section 6 that made meth so compelling can also make a second dissociative substance appealing. The route of administration matters significantly here: IV ketamine and esketamine (Spravato, intranasal, FDA-approved for treatment-resistant depression — an off-label use). if applied to stimulant use disorder) have far higher and more reliable bioavailability than oral ketamine, including troches — oral routes lose significant drug to first-pass liver metabolism, producing a less predictable effect. In this early, still-actively-repairing window, the more reliable IV or intranasal routes are generally considered the more clinically worthwhile option if this path is pursued at all; oral ketamine is more commonly discussed as a later-stage option, closer to the one-year mark once the acute dopamine and glutamate repair work in Section 3 is substantially further along. This is a conversation for a psychiatrist familiar with Both stimulant use disorder and ketamine's research base are not a self-directed addition to this stack.

Tier 2 — Medical and Psychiatric Support

- There is no FDA-approved medication specifically for methamphetamine withdrawal — unlike the benzo or SSRI guides, this tier is about monitoring and support rather than a pharmacological bridge.
- Una evaluación psiquiátrica durante la fase de crisis es apropiada si la depresión es grave, dada la ventana real de riesgo de suicidio en los días 1 al 5; esto no es una reacción exagerada, sino una respuesta proporcional a un período de riesgo documentado.
- Si un médico prescribe algún medicamento (a veces se usa bupropión fuera de indicación para el trastorno por uso de estimulantes), informe sobre el estado de CYP2D6/CYP2B6 si se conoce, según la Sección 6.

Tier 2.5 — 'Ghost Scaffolding': Psychiatrist-Supervised Stimulant Stabilization

Detoxification with medication:

* This deserves its own honest treatment rather than a single bullet, because it's a real approach that exists in specialist addiction medicine, and it's also genuinely easy to misunderstand as 'just get a prescription for the same kind of drug.' It is not that, when done correctly — but the distinction matters enough to walk through carefully.

⚠ CAUTION

This is not a self-directed strategy. It requires a psychiatrist or addiction medicine specialist who is actively managing the decision, monitoring for misuse risk, and treating it as a time-limited clinical tool — not an open-ended replacement. Pursuing a stimulant prescription informally, or from a general prescriber unaware of the full history, is a materially different and much riskier thing than what's described below.

The clinical logic: the Gray and Slow Return phases in Section 2 produce real, measurable executive-function impairment — attention, working memory, task initiation — that can closely resemble ADHD, whether or not ADHD was present before meth use began. In some specialist settings (this exists as documented practice in some countries' addiction medicine programs, sometimes called psychostimulant substitution or agonist-style stabilization), a carefully supervised, long-acting prescription stimulant — extended-release amphetamine salts (Adderall XR) or similar — is used short-term specifically to bridge that executive-function gap while the dopamine system in Section 3 does its slower underlying repair work, in the similar to how using buprenorphine can stabilise a person who is dependent on opioids, instead of letting their body try to recover completely without any support.

Wat dit is.	Wat dit niet is.
A time-limited, specialist-supervised clinical tool designed to address the specific executive function deficits observed during the Gray/Slow Return phases of Section 2.	Een groen licht om de "crash" zelf te behandelen met elk stimulerend middel, voorgeschreven of niet.
Used alongside, not instead of, the Tier 1 and Tier 3 work in this section.	En remplacement des exercices, du sommeil et du travail de neuroplasticité qui favorisent réellement la récupération des récepteurs D2 dans la section 3.
Evidenza limitata e oggetto di dibattito nell'ambito della medicina delle dipendenze – livello di evidenza: emergente, non consolidato.	A well-established, first-line standard of care —most addiction specialists still approach this with caution, or not at all.

BEING HEARD

If the fog and flatness of early recovery are making you wonder whether you actually have ADHD, or always did — that's a legitimate question worth raising with a psychiatrist, not something to dismiss as 'just withdrawal talking.' Sometimes it is

withdrawal. Sometimes a real, previously undiagnosed ADHD was part of what made stimulants so compelling in the first place, per Section 6's phenotyping questions. A specialist can actually help you tell the difference, which is exactly the conversation 'ghost scaffolding' is meant to open, not replace.

Tier 3 — Nervous System and Neuroplasticity Support (The Repair Signal)

- Esercizio: l'intervento più importante di questo intero protocollo. Il BDNF, il segnale di riparazione per gli assoni dopaminergici danneggiati documentati nella Sezione 2 e nella Sezione 3, viene prodotto principalmente attraverso l'attività fisica. Inizia non appena possibile, anche brevemente, e aumenta gradualmente fino a raggiungere 30-45 minuti di intensità moderata-alta quasi tutti i giorni.
- Sommeil : 7 à 9 heures, respectez un horaire régulier ; la synthèse du BDNF et la consolidation de la mémoire atteignent leur maximum pendant le sommeil, ce qui signifie qu'un sommeil irrégulier ralentit directement le processus de réparation décrit dans la section 3.
- Luminosidade da manhã — 20+ minutos ao ar livre, idealmente nas primeiras duas horas após acordar, para alinhar o ritmo circadiano e melhorar o humor.
- A novel, challenging learning activity—spending 30+ minutes each day on something that is actively difficult (like playing an instrument, learning a language, or mastering a complex skill)—promotes neuroplasticity more effectively than passive activities.
- Strutturata routine quotidiana e riprogettazione dell'ambiente: eliminare o modificare i fattori scatenanti legati ai luoghi di utilizzo, alle persone e agli oggetti.

Section 9 — On Stigma

Stigma can be a significant barrier to accessing mental health care. It can lead to feelings of shame, isolation, and discrimination, which can make it difficult for people to seek help.

There are many different types of stigma, including:

*** Public stigma: This is the negative attitudes and beliefs that the general public holds about people with mental illness.**
*** Self-stigma: This is the internalized negative beliefs that people with mental illness hold about themselves.**
*** Structural stigma: This is the policies and practices that discriminate against people with mental illness.**

Stigma can have a number of negative consequences for people with mental illness, including:

*** Reduced access to care: People who experience stigma may be less likely to seek help for their mental health problems.**
*** Social isolation: Stigma can lead to feelings of shame and isolation, which can make it difficult for people to connect with others.**
*** Discrimination: People with mental illness may face discrimination in employment, housing, and other areas of life.**

There are a number of things that can be done to reduce stigma, including:

*** Education: Educating the public about mental illness can help to dispel myths and misconceptions.**
*** Contact-based interventions: These interventions involve people with mental illness sharing their stories with others.**
*** Advocacy: Advocating for policies and practices that promote inclusion and equality for people with mental illness.**

It is important to remember that stigma is not a personal failing. It is a social problem that can be addressed through education, contact-based interventions, and advocacy.

Methamphetamine carries a specific, heavier weight of social stigma than most other substances covered in this library — the 'tweaker' stereotype, assumptions about criminality or moral failure, and a level of disgust in how the drug and its users get talked about publicly that alcohol or even opioid dependence often doesn't carry to the same degree. This is worth naming directly rather than leaving unspoken, because it has a real, documented practical effect: people with methamphetamine use disorder report — and research on healthcare-provider bias confirms — receiving measurably worse quality care, less thorough pain management, and more dismissive treatment. from medical providers than people with other substance use disorders, even when presenting with the same symptoms.

BEING HEARD

If you've felt like you had to brace yourself before telling a doctor, a dentist, or an ER what was actually going on — that instinct isn't paranoia. It's a reasonable response to a real pattern. It doesn't mean every provider will treat you that way, and it doesn't mean you should hide the information that keeps you safe (per Section 0 and Section 5's psychosis-risk overlap, an accurate history matters for your own care) — but the caution you're carrying into that conversation is not an overreaction to nothing.

- If a healthcare provider's tone changes significantly once methamphetamine use is disclosed, it's important to address this directly with them ("I want to ensure that this history doesn't affect the quality of care I receive"). If this doesn't resolve the issue, you have the right to seek care from a different provider.
- Specialists in addiction medicine and clinics focused on harm reduction often exhibit less of this bias compared to general practices or emergency departments. It's worthwhile seeking them out specifically for ongoing care when possible.
- The stigma relates to the drug's reputation, not to you personally. The dopamine system rebuilding process described in Section 3 is unaffected by any assumptions people might make about your character while it's happening.

Section 10 — Emergency Recognition, Restated

⚠ CAUTION

Call 13 11 14 or 000 immediately for: suicidal thoughts or a specific plan, chest pain or irregular heartbeat, sudden severe headache or stroke signs (facial drooping, arm weakness, slurred speech), psychosis (paranoid delusions, hallucinations), or extreme

agitation with loss of behavioral control. For the crash symptoms alone — exhaustion, hypersomnia, depression, cravings, cognitive fog — these are expected per Section 2, but a support contact and, if severe, a same-day medical or psychiatric evaluation are still appropriate.

Section 11 — Full Protocol Cross-Reference Map

Reference	What It Offers Here
Recovery Kernel Meth/Stimulant Guide, Section 3 (The Detox Protocol)	The full source timeline this guide's Section 2 is built from, plus the complete supplement dosing and timing detail behind Section 8's Tier 1.
Recovery Kernel Compendium, Section 16.4 (Receptor Recovery — The Evidence)	Volkow et al. och Heinz et al. hänvisningarna bakom avsnitt 3:s tidslinje på 12–17 månader, samt jämförande data för återhämtning av alkohol- och kokainreceptorer.
Recovery Kernel Compendium, Protocol 01 (Shelter)	The crisis-stabilization framework, as discussed in Section 2, applies to both the "crash" and "pit" phases.
Recovery Kernel Compendium, Protocol 05 (Environmental Redesign)	La eliminación de desencadenantes y la modificación del entorno a los que se hace referencia en la Sección 8, Nivel 3: identificación y alteración de personas, lugares y objetos asociados al consumo.
Recovery Kernel Compendium, Protocol 06 (Daily Structure)	Il quadro di riferimento delle routine costanti è appropriato a partire dalle settimane 2-4 della Sezione 2, una volta che l'acuta fase critica è passata.
Recovery Kernel Compendium, Protocol 20 (Identity Reconstruction) and C-28 ('The Recast').	Appropriate starting around Months 1-3 in Section 2 — not earlier. Building an identity and a set of genuine, high-intensity outlets suited to the Warrior COMT profile in Section 6, rather than one organised around either the drug or its absence.
Recovery Kernel Compendium, Section 16 (Neuroplasticity Architecture)	The full 90-day rebuilding framework referenced in Section 3: * Exercise, sleep, learning, and sunlight protocols in complete detail, including the weekly structure behind Section 8's Tier 3.
Recovery Kernel Chemsex Guide	Se l'uso di metanfetamina si è verificato nel contesto del chemsex, la tabella dei tempi di recupero combinata per GHB/metamfetamina/mephedrone e lo schema integrativo unificato affrontano il quadro della polidrugdipendenza. Questa guida copre solo la metanfetamina singolarmente.
Recovery Kernel: Complex Trauma and the Substance Connection.	Per this guide's own Section 3 (Substance-Trauma Co-Axis): chronic hypervigilance channeled into forced achievement is a documented trauma-driven pathway into stimulant use specifically — worth reading alongside this guide if that pattern applies.

Section 12 — Closing

SANCTUARY

The dopamine system that methamphetamine hijacks is not permanently broken. The data is specific and real: measurable recovery within weeks, substantial structural recovery within a year to a year and a half, and a 90-day window where deliberate exercise, sleep, and novel challenge do more rebuilding work than any other point in the process. The misery of the crash was never a sign that something had gone wrong. It was, and is, the signal that repair had already begun.

END OF GUIDE — DISCONTINUANCE PROTOCOL: METHAMPHETAMINE

Harm Reduction Resource — Not medical advice — Not a substitute for emergency care.

Crisis Resources: 988 (Suicide & Crisis Lifeline, call or text) | Crisis Text: HOME to 741741 | SAMHSA: 1-800-662-4357